

Message

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Sent: 11/17/2017 12:44:29 AM
To: Keller, Kaitlin [keller.kaitlin@epa.gov]
CC: Beck, Nancy [beck.nancy@epa.gov]; Bertrand, Charlotte [Bertrand.Charlotte@epa.gov]
Subject: questions

Kaitlin

Here are some questions for our OPP colleagues:

- Part of the evaluation of the epidemiology studies was how well exposure was measured. What call did the Columbia studies get? If more than low, why did the recent SAP defer on using exposure information from this study in a quantitative assessment?
- How were blood leads associated with the neurological outcomes independent of chlorpyrifos?
- Many of these epidemiology studies are observational. What is the hypothesis being considered from these statistically significant associations?
- Has this hypothesis been tested in definitive studies?
- If the chlorpyrifos metabolite is primarily made in the liver, and if this metabolite irreversibly binds to cholinesterase, then how does any of it get to the brain at concentrations in the blood that do not inhibit cholinesterase (~0.03 mg/kg-day from slide 23 of PBPK briefing on 11-15)?
- Experimental results in rats suggest that the fetus is less susceptible to cholinesterase inhibition than the dam. So why would human fetuses be more susceptible than their mothers?

Cheers!

Michael...

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